IN THE UNITED STATES DISTRICT COURT

FOR THE DISTRICT OF DELAWARE

BAYER AG and BAYER CORPORATION, Plaintiffs, v. HOUSEY PHARMACEUTICALS, INC., Defendant.	)	Civ. No. 01-148-SLR

O R D E R

At Wilmington, this 4th day of December, 2003, for the reasons stated in the opinion issued this same day;

IT IS ORDERED the clerk shall enter judgment in favor of the plaintiffs Bayer AG and Bayer Corporation and against defendant Housey Pharmaceuticals, Inc.

United States District Judge

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BAYER AG and BAYER CORPORATION, Plaintiffs, v. HOUSEY PHARMACEUTICALS, INC., Defendant.	)	Civ. No. 01-148-SLR

Rudolf E. Hutz, Esquire, Jeffery B. Bove, Esquire and Mary W. Bourke, Esquire of Connolly Bove Lodge & Hutz LLP, Wilmington, Delaware. Counsel for Plaintiff. Of Counsel: Thomas F. Poché, Esquire, of Connolly Bove Lodge & Hutz LLP, Wilmington, Delaware.

M. Duncan Grant, Esquire and Joseph S. Naylor, Esquire of Pepper, Hamilton LLP, Wilmington Delaware. Counsel for Defendant. Of Counsel: Rolf Stadheim, Esquire, Joseph A. Grear, Esquire and George C. Summerfield, Esquire of Stadheim & Grear, LTD., Chicago, Illinois.

OPINION

Dated: December 4, 2003
Wilmington, Delaware

ROBINSON, Chief Judge

I. INTRODUCTION

The court tried the single issue of inequitable conduct by Housey Pharmaceuticals, Inc.[1] ("Housey") in a four day trial, commencing on December 3, 2002 and ending December 9, 2002. The court has jurisdiction over this action pursuant to 28 U.S.C. §§ 1331, 1338(a) and 2201(a). Having considered the documentary evidence and testimony, the court makes the following findings of fact and conclusions of law pursuant to Fed. R. Civ. P. 52(a).

II. FINDINGS OF FACT

A. Procedural History

1. Bayer AG is a holding corporation organized and existing under the laws of the Republic of Germany and having its principal place of business in Leverkusen, Germany. Bayer Corporation is a corporation organized under the laws of the State of Indiana, with its principal place of business in Pittsburgh, Pennsylvania. Bayer AG and Bayer Corporation, (jointly referred to as "Bayer") are principally engaged in the development and manufacture of chemicals and pharmaceuticals.

2. Housey is a corporation organized and existing under the laws of Delaware and having its principal place of business in Troy, Michigan. Housey engages in the research,

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[1]Housey changed its name from ICT Pharmaceuticals, Inc. (D.I. 38)

development, and licensing of pharmaceutical products.

3. Bayer filed this action on March 6, 2001 seeking a declaratory judgment of invalidity, noninfringement, and unenforceability due to inequitable conduct against four patents assigned to defendant Housey by Dr. Gerard M. Housey, M.D., Ph.D. (D.I. 1) The patents in suit are U.S. Patent Nos. 4,980,281 (issued Dec. 25, 1990, the "'281 patent"), 5,266,464 (issued Nov. 30, 1993, the "'464 patent"), 5,688,655 (issued Nov. 18, 1997, the "'655 patent"), and 5,877,007 (issued March 2, 1999, the "'007 patent") (collectively, the "Housey patents").

4. Housey counterclaimed against Bayer alleging direct infringement, contributory infringement, inducement of others to infringe, and infringement pursuant to 25 U.S.C. § 271(g). (D.I. 5)

5. The court dismissed Housey's claim for infringement under § 271(g) on the basis that a "plain reading of the statute ... addresses only products derived from patented manufacturing processes." Bayer AG v. Housey Pharms., Inc., 169 F. Supp. 2d 328, 330 (D. Del. 2001), aff'd 340 F.3d 1367 (Fed. Cir. 2003).

6. On November 12, 2002, the court entered an order construing the claims of the Housey patents. (D.I. 254) That order was certified pursuant to Rule 54 and is on appeal to the Federal Circuit.

7. The parties subsequently entered into a stipulation, contingent upon claim construction, that the Housey patents are invalid and unenforceable. (D.I. 269) Notwithstanding, the parties sought to proceeed with a bench trial on the issue of inequitable conduct.

B. Technological Background

8. The Housey patents, each entitled "Method of Screening for Protein Inhibitors and Activators," generally relate to research methods used by pharmaceutical companies for discovering drugs. (D.I. 1)

9. Pharmacological research assays may be classified into three general categories. The first are biochemical assays, in which whole cells are broken down and proteins are separated, purified and test substances applied. (D.I. 280 at 52-54) Biochemical assays are the primary starting point for most pharmacological research. (Id.) The second approach, cellular assays, in contrast, involve the application of test substances to whole cells to determine the cellular response. (Id. at 55) Finally, pharmacological researchers will employ the use of animal studies and eventually human studies in their development of new pharmacological products. (Id. at 53)

10. Cellular assays can test for a variety of cellular responses to tested substances, from simple toxicity to various phenotypic responses such as an increase in the rate of growth.

(Id. at 55-56)

11. The Housey patents are directed to a cellular assay described as a "method for screening for substances which specifically inhibit or activate a particular protein affecting the cultural or morphological characteristics of the cell expressing the protein." (JTX 2, '281 patent, col. 1, ll. 10-13) The expression of the "particular protein" (referred to as the "protein of interest") results in a change in one or more identifiable characteristics of the cells expressing it. The patented methods enable researchers to screen substances for active compounds that indicate a potential for development as pharmaceuticals through the use of a whole cell assay. (D.I. 280 at 67)

12. The claimed method states that a cell line is produced that is characterized by a higher production of the protein of interest relative to an original cell line. (Col. 4, ll. 40-50) By applying substances ("agents") to both cell lines, it is possible to determine whether the agent is an activator or inhibitor of protein activity.[2]

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[2]An activator or inhibitor of a protein was defined by the court to mean "a substance that has a greater effect on the phenotype of cells that express the protein of interest at a higher level than on the phenotype of cells that express the protein of interest at a lower level or not at all." (D.I. 248 at 2) In so construing, the court rejected the claim limitation proposed by Housey that an inhibitor or activator must "directly bind" with the target protein.

13. At the time of Dr. Housey's invention, it was believed that whole cell assays could not be used to identify a specific activator or inhibitor of a particular protein in a cell. (D.I. 280 at 70) This is because within a whole cell there are multiple protein enzymes which may interact with the test substance, or each other, precluding a conclusion as to whether a specific protein is activated or inhibited. (D.I. 280 at 67)

14. As embodied in claim 1 of the '281 patent, the method requires two cell lines, one cell line which overexpresses a given target protein and shows a phenotypic response that is evoked by the target protein, relative to the second cell line. (Col. 24, ll. 51-53; D.I. 280 at 60-61)

15. The phenotypic response must be observable prior to the addition of test substances to the cell. (D.I. 254) Substances are then incubated with, or added to, these cells, and the phenotype of the cells is studied for further changes. (Col. 24, ll.59-63; D.I. 280 at 61)

16. Table 2 of the Housey patents contains numerical data that illustrate the invention as applied to activators. (Col. 20, ll. 28-44) Table 3 of the Housey patents contain numerical data that show the invention as applied to inhibitors. (Col. 20, ll. 46-70 to col. 21, ll. 1-7) Both of these tables utilize protein kinase C ("PKC") overexpressing cell line. (D.I.

280 at 63) Dr. Housey specifically relied on this data to overcome the examiner's rejection of the claims in the '281 patent. (JTX 6 at B5000111)

C. Events Preceding the Submission of the '281 patent

17. Dr. I. Bernard Weinstein is an accomplished professor and researcher at Columbia University. He has co-authored over 550 publications and has been the recipient of numerous awards and research grants. (PTX 228) Dr. Weinstein joined the Columbia University faculty to pursue a career in clinical oncology. (D.I. 281 at 595, 597, 600)

18. Dr. Weinstein's work has focused on the development of cancer fighting drugs, including the identification of tamoxifen as an inhibitor of PKC. (Id. at 600-01)

19. Dr. Weinstein's research has involved the screening of compounds using cell-based assays. In 1978, he was involved in introducing genes into cells, altering their phenotype and showing that exposure of these cells to a drug would give exaggerated or altered responses. (Id. at 601)

20. In 1984 and 1986, Dr. Weinstein co-authored papers with Dr. Wendy Hsiao on the use of c-Harvey-ras ("c-Ha-ras") oncongene overexpressing cells as a screening assay for new tumor promoters. (PTX 312-13) These experiments showed that c-Ha-ras expression in cells induces a phenotypic change in the cells, and

that this method may be used as a tool to screen for compounds that would modulate this change.

21. On January 20, 1984, Dr. Weinstein applied for a grant that focused on tumor promotion and PKC (the "1984 Grant"). (D.I. 281 at 627-28; PTX 113A) That application included the plan to clone the PKC gene, overexpress the protein in the cells, and test those cells with compounds. The 1984 Grant application suggested that cloning the PKC gene and its overproduction in cells would phenotypically alter or transform the cells. (D.I. 281 at 669-71; PTX 113A at C000798)

22. The 1984 Grant application states that these engineered cells would be analyzed for altered responsiveness to substances (TPC or teleocidin) and it refers to Dr. Weinstein's interest in developing new methods of detecting tumor promoters and strategies for cancer treatment. (D.I. 281 at 673-74; PTX 113A at C000751-799)

23. The 1984 Grant application refers to establishing screening tests for tumor promoters and tumor blocking agents based on the information that was expected to be provided under the Grant. (D.I. 281 at 674-75; PTX 113A at C000771)

24. Dr. Weinstein is and was, during the relevant time period, responsible for overseeing grants and for setting the direction of research at the Columbia University laboratory (the "Weinstein laboratory"). (D.I. 281 at 604; D.I. 283 at 785, 816,

845) He routinely provides guidance and instruction to graduate students as part of these responsibilities. (D.I. 281 at 605; D.I. 283 at 785, 816, 845)

25. Dr. Housey is the sole named inventor of the Housey patents and currently serves as President and CEO of Housey, the defendant and patent assignee.

26. Dr. Housey attended the University of Michigan from 1977 to 1981 where, as an undergraduate student, he developed research skills with respect to standard laboratory methods, tissue culture techniques, and in cellular assay systems. (D.I. 283 at 894-96)

27. Due to his advanced skills, Dr. Housey received special authorization to enroll in graduate and medical school courses before the completion of his undergraduate education. (Id. at 892-93) As a result, he developed experience with cellular assays prior to joining the Weinstein laboratory. (Id. at 892, 902; DTX 5)

28. Dr. Housey performed his graduate work in the laboratory of Dr. Weinstein at Columbia University between 1982 and 1988. (D.I. 282 at 715, 717-18; D.I. 283 at 892, 902) Dr. Housey was described as a talented researcher in the Weinstein laboratory. (DTX 22; DTX 125; D.I. 281 at 470-71; D.I. 282 at 715, 717-19)

29. While in the Weinstein laboratory, Dr. Housey

worked extensively with PKC. (D.I. 290 at 201; D.I. 283 at 1020-21; DTX 13 at 1) In 1990, Dr. Weinstein described Dr. Housey as having carried out PKC studies "almost single handedly." (DTX 22)

30. Over a three and a half year period, while a graduate student, Dr. Housey cloned and sequenced the PKC gene as his thesis project. (D.I. 283 at 1020)

31. Following the successful cloning of PKC, it was determined that PKC is a multigene family of enzymes that exist in multiple isoforms, which are distinct but related species. (PTX 501 at 343; D.I. 280 at 31-32) The specific isoform cloned by Dr. Housey was named PKC beta 1. The cloning project was performed under the 1984 Grant that Dr. Weinstein had secured for the laboratory.

32. The PKC gene subsequently was the subject of a patent application filed in July 1987, of which Dr. Housey, Weinstein, Mark Johnson, and Catherine O'Brian were named as co-inventors. (PTX 114 at B5100004) Each of these inventors assigned their rights in the patent to Columbia.

33. Dr. Housey's PKC work culminated in the preparation, submission, and defense of his thesis in November 1987, for which Dr. Housey was awarded his Ph.D. from Columbia. (D.I. 282 at 722; DTX 13)

D. Research Reported in the Cell Paper

34. In February 1988, Dr. Housey published in the

scholarly journal Cell, an article entitled, Gerard M. Housey et al., Overproduction of Protein Kinase C Causes Disordered Growth Control in Rat Fibroblasts, 52 Cell 343 (1988) (hereinafter "Cell Paper"), which was excerpted nearly verbatim from the work Housey reported in Chapter 4 of his Ph.D. thesis. (PTX 601; DTX 13) The Cell Paper was directed to the overexpression of PKC in rat fibroblast cells. (D.I. 280 at 242; DTX 13 at C00084-128) Dr. Housey is the first named author on the Cell Paper, along with several other researchers from Weinstein's laboratory, including Dr. Weinstein.[3]

35. The work of other researchers in the Weinstein laboratory was reported in the Cell Paper, including that of Dr. Robert Krauss, Dr. Wendy Hsiao, Dr. Paul Kirschmeier, and Dr. Mark Johnson. (PTX 601) The Cell Paper discloses, inter alia, the PKC cloning and the sequencing work of Dr. Housey, the cell line generation work of Dr. Johnson, and the cellular analysis of Dr. Hsiao. In a subsequent invention report, Dr. Housey

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[3]It is the practice in the scholarly publication of scientific research findings that when multiple authors are cited, each of these authors is considered to have contributed in some way to the production of the findings. (D.I. 281 at 296) It is generally the case that the first named co-author will be the individual most responsible for generating the research. (D.I. 280 at 138) It is also generally the practice that the co-author listed last will be the "principal investigator," the person in a research laboratory who "sets the direction", obtains grants, and provides general oversight. (Id. at 38, 138)

described the Cell Paper as "definitive" and as providing "much of the framework for this patent application." (PTX 21, at 38383)

36. The PKC overexpressing cell lines, referenced as Rat 6 cell lines in the Cell Paper and Housey patents, were developed by Dr. Mark Johnson, a post doctoral researcher and co-inventor on the PKC gene application. Dr. Johnson used the pMV-7 expression vector[4] containing the PKC gene to generate the PKC overexpressing lines. (JTX 3; D.I. 283 at 940)

37. The Cell Paper reports on Dr. Hsiao's work with cellular assays showing that PKC beta 1 overexpressing Rat 6 cells displayed altered growth rates and acquired the ability to grow in soft agar. (D.I. 283 at 800; PTX 64 at WH00010-26, 39, 42, 45-48, 58, 65) Dr. Hsiao demonstrated that 12-O-tetra decanoyl phorbol-13-acetate ("TPA"), a known tumor promoter, increased the growth rate of these PKC beta 1 overexpressing cells and increased their ability to grow in soft agar. (D.I. 283 at 805-07; PTX 64 at WH00048, 56, 59). These results were described in the Cell Paper and reported in figures 4,5 and 6,

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[4]An expression vector is a tool needed to genetically alter and overproduce a protein of interest. (D.I. 280 at 279-80) The pMV-7 vector employed by Dr. Johnson was developed by Dr. Paul Kirschmeier, a post doctoral researcher in the Weinstein laboratory, and it was the first retroviral vector used to express cloned genes in genetically engineered cell lines. (JTX 3; D.I. 282 at 626; D.I. 281 at 461)

and table 2 of that paper without direct attribution to Dr. Hsiao. (PTX 601, at 349)

38. Figure 1 in the Cell Paper reports and illustrates the nucleotide sequence, pMV7-PKC expression vector construction, and the cell generation strategy utilized by Dr. Housey. (PTX 601 at fig. 1)

39. Figure 2 in the Cell Paper is an audiograph reporting the results of the purification and autophosphorylation of PKC. (Id. at fig. 2) This experiment provided independent evidence of the "presence of a high level of intact PKC molecules in these cell lines." (Id. at 345) The phosphorylated bands of the engineered cells when compared to a control line reflect the presence of PKC activity. (Id.)

40. Figure 3 in the Cell Paper reports the results of a Northern Blot hybridization analysis. (Id. at fig. 3) This analysis showed that those cell lines containing elevated levels of PKC also contained a prominent 6.6kb RNA species, which corresponds to the "predicted size for an mRNA transcript that initiates in the 5' LTR and terminates in the 3' LTR of the pMV7-PKC construct". (Id. at 347)

41. Figure 4 of the Cell Paper reports the morphological responses of the cell lines to phorbol ester treatment. (Id. at fig. 4) In this experiment, certain PKC overexpressing cell lines and a control cell line were treated

with TPA. PKC overexpressing cell lines displayed an enhanced morphological response to TPA in comparison to the control cell line.

42. Figure 5 of the Cell Paper reports on the study of postconfluence foci formation in the cell lines. (Id. at fig. 5) In this experiment, "monolayer cultures were maintained at postconfluence for an extended period of time (28 days), with media changes every 3 days in the absence of TPA." (Id. at 349-50. The researchers determined that the control line "remained a fairly uniform monolayer" whereas one of the engineered cell lines "developed numerous dense foci ... and ... numerous cells with a highly vacuolated cytoplasm." (Id. at 350) The paper postulated that these morphological changes suggested a physiologic change, rather than a genetic change.

43. Figure 6 of the Cell Paper illustrates the growth in soft agar of the cell lines. (Id. at fig. 6) In rodent cells, the ability to have anchorage-independent growth, such as in soft-agar, often correlates with tumorigenicity. (Id. at 350) The researchers found that the PKC expressing cell lines formed numerous small colonies when grown in soft agar, whereas the control cells persisted as single cells. This colony growth was enhanced by the addition of TPA to the agar medium. The Cell Paper concluded, therefore, that "the overproduction of PKC is associated with the acquisition of anchorage-independent growth

in Rat 6 cells." (Id.)

44. On February 10, 1988, two days prior to the Cell paper's publication, Dr. Housey filed the '281 patent with the U.S. Patent and Trademark Office.

45. Dr. Housey testified that at the time of the patent application, he believed his invention was directed toward an assay that utilized a "whole cell as a tool to identify inhibitors or activators of a specific target protein functioning in a particular cell environment." (D.I. 283 at 1004) Dr. Housey testified that he believed his method to involve: (1) identifying a phenotypic response associated with a specific target protein; and (2) utilizing that phenotypic response to identify agents which activate or inhibit the target protein by binding to that target protein.[5] (Id.)

46. Dr. Housey did not believe the Cell Paper taught the invention as Dr. Housey understood it. (Id. at 1012-13) He, however, did disclose the Cell Paper to the PTO. (D.I. 281 at 543-44)

E. Research Reported in Housey Patents

47. The '281 patent describes several examples of the

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[5]Dr. Housey testified that his understanding of his invention is different from the court's construction of the claims in the Housey patents. In particular, the court has construed the claims to not limit an "activator or inhibitor" of a protein to be one that "binds" with the protein.

invention, results of which are reported in figures and tables in the application. Nearly each of these are identical to experiments reported in the Cell Paper.[6]

48. In table 1(a) of the '281 patent, Dr. Housey reports the effect on PKC of infecting Rat 6 cells with pMV7 or pMV-7PKCbeta 1. (Col. 19, ll. 15-34) These identical results are reported as table 1 in the Cell Paper. See Cell Paper, supra, at 346 tbl. 1.

49. In table 1(b) of the '281 patent, Dr. Housey reports the effect on PKC of infecting NIH-3T3 cell lines with pMV7 or pMV7-PKCbeta1. (Col. 19, ll. 37-58) Table 1(b) is used as an example of the method cell types other than the Rat 6 cell line. (Col. 5, line 66 to col. 6, line 1)

50. In table 1(c) of the '281 patent, Dr. Housey reports the effect on PKC of infecting C3H-10T1/2 cell lines with pMV7 or pMV7-PKCbeta 1.[7] (Col. 19, ll. 59-70 to col. 20, ll. 1-

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[6]Compare '281 patent, fig. 1 with Cell Paper, supra, fig. 1(a); and '281 patent, fig. 2 with Cell Paper, supra, fig. 2; and '281 patent, fig. 3 with Cell Paper, supra, fig. 3; and '281 patent, fig. 4 with Cell Paper, supra, fig. 1(a); and '281 patent, fig. 1 with Cell Paper, supra, fig. 4; and '281 patent, fig. 6 with Cell Paper, supra, fig. 5; and '281 patent, fig. 7 with Cell Paper, supra, fig. 6; and '281 patent, tbl. 1(1) with Cell Paper, supra, tbl. 1; and '281 patent, tbl. 2 with Cell Paper, supra, tbl. 2. See also Joint Pre-Trial Order 11/18/2001. (D.I. 270)

[7]The research reported in table 1(c) had also been performed by Dr. Robert Krauss, a post doctoral researcher in the Weinstein laboratory. Dr. Krauss created a PKC overexpressing cell line

25) Table 1(c) is relied on by Dr. Housey as evidence of another example of the successful use of the invention. (Col. 5, line 66 to col. 6, line 1)

51. In table 2 of the '281 patent, Dr. Housey reports the effect on growth properties of Rat 6 cell lines that overproduce PKC and their response to TPA[8] treatment. (Col. 20, ll. 27-43) The identical results are reported in table 2 of the Cell Paper. See Cell Paper, supra, at 349 tbl. 2. (D.I. 270) It is not disputed that table 2 of the Cell Paper is the product of Dr. Hsaio's research. (D.I. 281 at 319)

52. The '281 patent asserts in the first person singular that "I also assayed these cell lines for their ability to form colonies in soft agar ..." (Col. 17, ll. 44-45)

53. In table 3 of the '281 patent, Dr. Housey reports the effect on growth in agar of PKC-overproducing cell lines in response to the introduction of growth inhibitors H-7 and

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utilizing C3H 10T1/2 cells ("10T1/2"). He showed that 10T1/2 PKC overexpressing cells displayed increased cell population, refractility and rounding relative to native lines. (D.I. 281 at 464) Dr. Krauss demonstrated that these cells formed foci when exposed to phorbol esters (D.I. 281 at 465) and, according to his laboratory notes, creation and characterization of these cell lines took approximately four months to complete. (D.I. 280 at 112-116; D.I. 281 at 466-67; PTX 78 at RK0000032, 58) In order to predict the phenotypic results of a protein overexpression, it is necessary to actually achieve the overexpression of that protein. (D.I. 283 at 844)

[8]Tumor promoting phorbol ester 12-O-tetradecanoylphorbol-13-acetate. (Col. 7, ll. 50-51)

tamoxifen. (Col. 20, ll. 46-70 to col. 21, ll. 1-7) Table 3 also demonstrates the effect of various inhibitors on the growth in soft agar of PKC beta 1 overexpressing Rat 6 cells. (D.I. 280 at 64, 169) This table was first reported by Dr. Housey in an invention report to his attorney. (PTX 75)

54. Table 3 is significant as it demonstrates how the invention works with inhibitors and it is a numerical representation of the working of the invention. (D.I. 283 at 876, 1013)

F. Corroboration of Dr. Housey's Research Results

55. The Weinstein laboratory consisted of one large and one small room in which twelve to eighteen scientists conducted research. (D.I. 281 at 603-604) It was common practice for scientists to share reagents, materials, and equipment. (D.I. 283 at 793-94, 945-46)

56. The Weinstein laboratory had a single incubator in which all cell culture research would have been conducted. (D.I. 283 at 793-94, 852-83, 945) At the relevant time period, single well tissue culture plates were the standard format for conducting cellular work in the Weinstein laboratory. (D.I. 281 at 435)

57. Multi-well plates, such as 24-well plates, were not used in the Weinstein laboratory as they could produce inaccurate results when using a microscope to count cell

colonies, due to optical issues involved with the multi-well plates. (D.I. 281 at 434-35; D.I. 283 at 853) Given the presence of a single incubator, it would have been likely, although not certain, that other researchers would have been aware of the use of 24-well plates. (D.I. 280 at 829-30; D.I. 283 at 796, 829-30, 853, 866)

58. Dr. Housey testified that the soft agar experiments underlying the data in table 3 of the '281 patent were conducted in duplicate using 24-well plates in the Weinstein laboratory. (D.I. 281 at 343) The 24-well plate provided for the ability to grow multiple data points in the same plate, simplifying the process. (Id.)

59. Dr. Housey was trained by Dr. White to use 24-well plates while an undergraduate student. (D.I. 283 at 893-94, 896, 899-902) Dr. White testified that 24-well plates were standard equipment in research laboratories by as early as 1982. (Id. at 898) Dr. White also testified that, given Dr. Housey's experience at Michigan, Dr. Housey could have performed the assays and obtain the results reported in his patents. (Id. at 902-03)

60. Primary data, with respect to standard laboratory research procedures, are contemporaneous records of experiments. (D.I. 280 at 82-84) Under standard practice at the time, researchers would maintain detailed contemporaneous notes related

to their observations. Dr. Housey's laboratory notes in general reflect this contemporaneous recording of primary data. (Id. at 85) Primary data is not limited to researcher notes and can include machine generated information, examples of which are autoradiograms, photomicrographs, simulation counter printouts. (Id. at 82-83)

61. Dr. Alan Fields, an expert witness on behalf of Bayer, testified that Dr. Housey's laboratory notes do not provide corroboration of Dr. Housey's testimony that he performed the work represented to the PTO. (Id. at 72) Dr. Fields testified that Dr. Housey appeared to maintain extensive primary notes with respect to other projects on which he had worked but, with respect to the Housey patents, there was an absence of primary data. (Id. at 85) In particular, Dr. Fields indicated that he was not able to identify any primary data related to the table 3 experiments. (Id. at 85, 129)

62. Dr. Housey testified that there may not be contemporaneous handwritten notes, as the manner in which he conducted the experiment rendered the need for handwritten notes unnecessary. (D.I. 281 at 339) Dr. Housey testified that he did not record primary data from these experiments by hand, but instead utilized a programmable calculator and attached printer to record the findings of certain experiments. (Id. at 353-53; D.I. 283 at 1024-25) There is no printed record, however,

reflecting this work.

63. Soft agar growth is calculated according a formula, which may programmed into a calculator to determine the growth in soft agar. (D.I. 280 at 170; D.I. 281 at 360-61)

64. Dr. Housey was known to be familiar with the use of electronic devices such as computers. (D.I. 283 at 862, 1001) Dr. Housey previously had used devices to record data and had found it more reliable to not use handwritten notes in certain kinds of experiments. (Id. at 899, 1024-25)

65. Several witnesses testified to the fact that they had no personal knowledge that Dr. Housey conducted the experiments. (Id. at 796, 852-53) Several witnesses testified that they did not think it would have been possible for Dr. Housey to conduct the experiments without their knowledge. (D.I. 282 at 771; D.I. 283 at 862, 939-40, 947-48) Certain witnesses also testified that they did not believe Dr. Housey had the skill necessary to conduct the experiments. (D.I. 281 at 462; D.I. 282 at 794; D.I. 283 at 853, 918-19)

66. Dr. Housey testified that he did not reveal to his colleagues the nature of his work pertaining to table 3. (D.I. 281 at 331)

G. Disclosure of Prior Art References to PTO

67. The use of cells for screening substances was well

known in the prior art at the time Dr. Housey filed the '281 patent. (Col. 1, ll. 16-65 to col. 2, 1-22) Prior art cellular assays, including cancer studies, looked for a variety of cellular changes, including reversion of the transformed phenotype. (Col. 2, ll. 9-22) Cellular assays were also helpful for cytotoxicity investigations, that is, looking at inhibition of cellular growth or metabolism to test substances for their ability to inhibit the growth of cells in soft agar, and to look for changes in morphology to determine the effectiveness of a given substance. (Col. 1, ll. 41-59)

68. Dr. Housey's initial attorney, Iver Cooper, submitted two papers as prior art to the PTO, the 1988 Cell Paper (PTX 601) and a paper by Julius, et al. (PTX 321)

69. **The Hsiao 1986 Reference.** The '281 patent refers to a paper by Dr. Wendy Hsiao as a source of the rat cell lines, the Rat 6 cell, used in Dr. Housey's research. (Col. 2, ll. 11-16, col. 5, 11. 56-58; D.I. 281 at 491; PTX 313, Hsiao, Wendy & Weinstein, I. Bernard, Oncongene-Induced Transformation of a Rat Embryo Fibroblast Cell Line is Enhanced by Tumor Promoters, 6 Molecular and Cellular Biology 1943 (June 1986) (hereinafter "Hsaio 1986")).

70. Hsiao 1986 describes a test cell line consisting of the Rat 6 cell line transfected with the mutant human c-Ha-ras activated oncongene. (D.I. 281 at 491-92) This evoked a

phenotypic response in the transfected cells, specifically, the formation of foci in culture. (Id.) Hsiao 1986 utilized untransfected Rat 6 cells as a control group. (Id.) Hsiao 1986 tested the effect of TPA on the c-Ha-ras overexpressing cells and the normal cells, and determined that TPA increased the phenotypic response (foci formation) of the c-Ha-ras-expressing cells, but did not affect the normal cells. (D.I. 281 at 492) Based on this research, the assay was proposed as a "simple screening test for detecting tumor promoters."[9] (PTX 313; D.I. 281 at 490-93)

71. The research reported in Hsiao 1986 did not involve screening for substances that directly bound with a target protein. (D.I. 281 at 510; D.I. 283 at 822-23)

72. The material relevance of the Hsiao 1986 reference is that it tends to show that the '281 patent specifications may not enable the direct interaction limitations found in the claims. (D.I. 280 at 66-70; Office Action dated Feb. 4, 2002 at 34126-30, PTX 617) In the Hsiao 1986 research, an agent was shown to interact indirectly with the c-Harvey-ras oncongene, an effect which undermines the '281 patent's claims that it can identify specific inhibitors or activators. (D.I. 281 at 493)

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[9]In a proceeding before the European Patent Office has admitted that Hsiao 1986 does not represent a complete screening process. (D.I. 294, ex. A)

73. Dr. Housey did not disclose the Hsiao 1986 reference as prior art to the PTO. Dr. Housey believed that there were critical differences between the Hsiao 1986 reference and his invention that rendered it nonmaterial. (D.I. 283 at 1048-49) In particular, Dr. Housey identified three limitations of his invention which were not present in Hsiao 1986: (1) "determining that a substance is an inhibitor or activator of a protein of interest;" (2) "defining a responsive change in a phenotypic characteristic in a cell by the functions of the target protein in the cell;" (3) "correlating the phenotypic response with the level of said protein functioning in the cell;" and (4) "realizing the value of ... and identifying that responsive change and using it to find unknown compounds, use it as a screen to find unknown or suspecting activators or inhibitors of a [protein of interest]." (D.I. 283 at 1048-49)

74. Dr. James Griffin, an expert testifying on behalf of Housey, further distinguished the Hsiao 1986 reference. (D.I. 283 at 966-70) That paper, according to Dr. Griffin, is directed at the effect of oncogene complementation and studying how "oncogenes work together to develop a full transforming ... phenotype in mammalian cell[s]."[10] (Id. at 969-70)

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[10]Oncogene complementation, which was first reported by a Dr. Robert Weinberg at the Massachusetts Institute of Technology, is a concept that suggests that certain weak oncogenes--oncogenes which individually may not have an affect on cells--when combined

75. The research of Hsiao et al. expanded upon this concept of complementation by testing whether certain tumor promoters, such as TPA, might have a complementary function, similar to the weak Ras oncogene, when introduced in cell assays.[11] (Id. at 968) Hsiao and her colleagues established that in some cells there was a complementary effect.

76. A second distinction made by Dr. Griffin between Hsiao's research and that of the '281 patent is that Hsiao 1986 involved TPA, a toxic chemical, not a drug screen. (Id. at 969)

77. **The Uehara 1985 Reference.** The '281 patent refers to a 1985 paper by Yoshimasa Uehara et al., as representative of a screening assay that depends upon a morphological alteration of the test cells. (Col. 2, ll. 9-22; PTX 375, Uehara, Yoshimasa et al., Screening of Agents which Convert 'Transformed Morphology' of Rous Sarcoma Virus-Infected Rat Kidney Cells to 'Normal Morphology': Identification of an Active Agent as Herbimycin and its Inhibition of Intracellular src Kinase, 76 Japanese J. of

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with another oncogene may have a greater affect. (D.I 283 at 967) Dr. Weinberg's research involved the oncogene's Ras and Myc. The significance of oncogene complementation is that the oncogenes complement "each other's activity work on separate pathways." (Id. at 967-68) Further, the concept teaches that, if a particular cell has the Ras oncogene, adding more Ras to the cell will not produce the same effect has adding the Myc oncogene. (Id. at 967)

[11]TPA is known to not bind directly to the Ras oncogene and does not activate nor inhibit its function. (Id. at 969)

Cancer Res. 672 (1985) (hereinafter "Uehara 1985")) The '281 patent criticizes morphology-based assays, such as described in the Uehara 1985 article, as difficult for practical reasons because they require an examination of the test cells under a microscope. (Col. 2, ll. 16-22)

78. The research reported in Uehara 1985 utilized cell lines stably infected with a temperature sensitive mutant of the Rous sarcoma virus[12] (RSV) designated ts/NRK. (D.I 281 at 483; PTX 375 at B2004448) These cell lines produce multiple proteins as a result of the Rous sarcoma infection. (D.I. 281 at 506; D.I. 283 at 962-63) When exposed to certain temperatures, the cell line will express the v-Src oncogene.[13] (D.I. 281 at 484; PTX 375 at B200448) An effect of the expression of the v-SRC oncogene in these cells is that the cells take on a transformed morphology relative to the normal uninfected cell line or to the same cell lines at an elevated temperature. (D.I. 281 at 485-86; D.I. 283 at 985-87)

79. The Uehara 1985 authors used these changes in morphology as a tool to screen for unknown compounds that would

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[12]Rous sarcoma refers to a group of retrovirus which has the capacity of transforming cells and making them cancerous. (D.I. 281 at 485)

[13]The v-SRC oncogene is the gene of the Rous sarcoma virus that has been identified as having the transforming capacity. (Id. at 485-86)

inhibit oncogene function. (D.I. 281 at 488; PTX 375 at B2004448) They determined that herbimycin, a known antibiotic, was capable of reversing the transformed phenotype of the v-Src expressing cells, but that it had no effect on uninfected cells. (D.I. 281 at 489)

80. Uehara and his colleagues also investigated the possible mechanisms for herbimycin's effect on the v-Src function in the infected cell line. During these experiments, they attempted to see whether herbimycin directly interacted with the v-Src protein. (D.I. 281 at 489; PTX 375 at B2004450) At that time, their research did not resolve whether the tested substances directly bound to the protein. (D.I. 281 at 489, 506)

81. The Uehara 1985 reference was not submitted to the PTO during the prosecution of the '281 patent. On December 13, 1989, Dr. Housey's original patent counsel, Iver Cooper, requested that Dr. Housey provide to him a copy of the Uehara reference.[14] (D.I. 280 at 253; PTX 24) Dr. Housey did not consider the Uehara 1985 reference to be material to his invention, as the article does not involve screening for substances that would bind with a particular target protein. (D.I. 281 at 506-07, 510, 511; D.I. 283 at 1047-52) In Dr. Housey's view, Uehara 1985 is substantially different from his

[14]On March 2, 1990, Kenyon & Kenyon replaced Cooper as prosecuting attorneys on the '281 patent. (D.I. at 248)

invention because Uehara does not focus on either a single protein of interest nor a defined responsive phenotypic change. (D.I. 283 at 1050) Instead, Uehara 1985 focuses more broadly on identifying substances which "inhibit or work or modulate oncogene" function. (Id.)

82. Dr. Griffin characterized the Uehara reference as being substantially different in two respects. First, Uehara used an intact virus to infect the cell line, rather than simply overexpressing a single protein in a cell. (D.I. 283 at 963) Second, Uehara's research involved temperature shifts to a temperature sensitive virus to control the activity of that virus during the experiment. (Id. at 964-65)

83. Even if Dr. Housey did not appreciate the materiality of the Uehara 1985 article at the time of his initial patent application, that reference was subsequently raised by two potential licensees, as early as 1995, in discussions with Dr. Housey concerning the potential licensing of the '271 and '464 patents. (D.I. 280 at 254-55)

H. Disclosure of Ownership and Inventorship Issues

84. Dr. Hsiao and Dr. Weinstein's prior work is cited in the Housey patent specifications. (D.I. 282 at 755-56, 758-59)

85. Dr. Weinstein believes that data contained in the Housey patents are the product of research performed in his

laboratory by researchers other than Dr. Housey. (D.I. 282 at 624) Moreover, Dr. Weinstein testified that he did not believe the data results contained in table 3 of the '281 patent to be possible. (Id. at 739) Dr. Weinstein testified, however, that if the Housey patents are construed as disclosing a method for identifying substances which directly bind or interact with a specific protein, he does not claim to be a co-inventor. (D.I. 282 at 774)

86. Dr. Hsiao believes that some of the work reflected in the Housey patents is appropriated from work she herself performed. (D.I. 283 at 799) Dr. Hsiao testified, however, that if the Housey patents are construed as disclosing a method for identifying substances which directly bind or interact with a specific protein, she does not claim to be a co-inventor. (D.I. 283 at 826)

87. Columbia University's policies related to its ownership interest in intellectual property developed in its facilities did not apply to Dr. Housey's work at the time Dr. Housey was enrolled.[15] (D.I. 281 at 588-89) Based upon this, and other facts, Dr. Housey received advice of counsel that Columbia University had no ownership interest in his inventions. (D.I. 281 at 531, 559; D.I. 280 at 291)

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[15]The University subsequently modified its policy to include work by students performed in its facilities. (Id. at 589)

88. In 1986, Dr. Housey incorporated a startup pharmaceutical company, Progenics Pharmaceuticals, Inc. ("Progenics"). (D.I. 281 at 564-65)

89. In support of Progenics, Dr. Housey actively engaged in negotiations with Columbia in an effort to license the PKC gene application, of which Dr. Housey was a co-inventor. Dr. Housey learned that any license would be nonexclusive and that any improvements would be subject to a grant-back provision. (D.I. 280 at 224; D.I. 281 at 384-85; PTX 32 at CD000438)

90. In February 1990, Dr. Housey separated from Progenics. (D.I. 280 at 268-69) In connection with a separation agreement, Dr. Housey acknowledged that "some of the individuals who have collaborated with Dr. Housey during his work as a graduate student at the Columbia Cancer Center, Columbia University itself, or the U.S. Government ... may attempt to claim some type of ownership or inventorship rights to the screening technology patents and applications on which Dr. Housey claims sole inventorship." (PTX 95)

91. On June 20, 1990, Kenyon & Kenyon provided an opinion letter to Dr. Housey in connection with his separation from Progenics. (PTX 71) The opinion letter stated that Columbia University did not have a valid basis to claim ownership in the Housey patents. (Id.)

92. In 1993, Columbia first learned of the '281 patent

from an attorney at Bristol Meyers. (D.I. 282 at 620-21) Columbia contacted Dr. Housey regarding the patents, under the belief that it may have an ownership interest. (D.I. 281 at 575; PTX 3)

93. In 1993, through an exchange of letters with Dr. Housey's attorneys, Columbia University asserted that it had an ownership claim in the Housey patents and possible inventorship claims on behalf of Dr. Weinstein. (PTX 3, 6, 9, 12, 13) These claims were subsequently dropped by Columbia.

III. CONCLUSIONS OF LAW

94. Bayer contends that the Housey patents are unenforceable because the applicant intentionally withheld material information during prosecution concerning the performance of certain experiments, prior art references, inventorship and ownership claims.

95. A patent applicant has a duty to prosecute his patent with candor, good faith and honesty. See Molins PLC v. Textron, Inc., 48 F.3d 1172, 1178 (Fed. Cir. 1995) ("Applicants for patents are required to prosecute patent applications in the PTO with candor, good faith, and honesty."). This duty is held jointly by the inventors, agents, attorneys, assigned and those otherwise involved in the preparation and prosecution of the patent application before the PTO. Id. at 1178 n.6.

96. Because the "duty of candor extends throughout the

patent's entire prosecution history ... a trial court may look beyond the final claims to their antecedents." Fox Indus., Inc. v. Structural Preservation Sys., Inc., 922 F.2d 801, 803-04 (Fed. Cir. 1991). "A breach of the duty of candor early in the prosecution may render unenforceable all claims which eventually issue from the same or a related application." Id.

97. Inequitable conduct consists of either an affirmative misrepresentation of a material fact, or the omission thereof, with the intent to deceive. Molins PLC, 48 F.3d at 1178. Under Federal Circuit precedent, a finding of inequitable conduct requires a two-step analysis. Perspective Biosystems v. Pharmacia Biotech, 225 F.3d 1315, 1318 (Fed. Cir. 2000). As an initial matter, the "court must determine whether the conduct meets a threshold level of materiality." Florida State University Board of Educ. v. American Bioscience, Inc., 333 F.3d 1330, 1343 (Fed. Cir. 2003). Then, the court must insure that the "threshold level of intent to mislead the PTO" is also present. Id. (citations omitted). Both materiality and intent to deceive are questions of fact which must be proven by clear and convincing evidence. Monon Corp., 239 F.3d at 1261 (citing Manville Sales Corp. v. Paramount Sys., Inc., 917 F.2d 544, 551 (Fed. Cir. 1990)).

98. Once both materiality and an intent to deceive are established, the court must weigh the evidence, in light of all

the circumstances, to "determine whether the applicant's conduct is so culpable that the patent should be held unenforceable." American Bioscience, 333 F.3d at 1343 (citations omitted).

99. Materiality, under Federal Circuit precedent, tests whether there is a "substantial likelihood that a reasonable examiner would have considered the information important in deciding whether to allow the application to issue as a patent."[16] Molins PLC, 48 F.3d at 1179. A reference is not considered material if it is not as relevant as that actually considered by the examiner or if it is merely cumulative of the information considered by the examiner. See id.

100. The materiality standard was derived, in part, from the PTO's previous disclosure rules. See 37 C.F.R. § 1.56(a) (1991). Dayco Prod. Inc. v. Total Containment, Inc., 329 F.3d 1358, 1363 (Fed. Cir. 2003). On March 16, 1992, the PTO amended § 1.56(a), adopting a narrower standard of materiality, defining materiality as information which "establishes either 'a prima facie case of unpatentability' or 'refutes, or is inconsistent with a position the applicant takes.'" Id. at 1364

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[16] "Information concealed from the PTO may be material even though it would not invalidate the patent. . . . As stated, the test for materiality is whether a reasonable examiner would have considered the information important, not whether the information would conclusively decide the issue of patentability." Li Second Family L.P. v. Toshiba Corp., 231 F.3d 1373, 1383 (Fed. Cir. 2000), cert. denied, 533 U.S. 929 (2001).

(quoting 37 C.F.R. § 1.56 (1992)).

101. The governing standard for materiality is the one in place when the pertinent events of the patent prosecution occurred. See CFMT, Inc. v. YieldUP Int'l Corp., 144 F. Supp.2d 305, 316 (D. Del. 2001). In the present case, three of the four patents were still pending at the time Rule 56 was adopted. The parties, in this case, agree that the pre-1992 standard applies.

102. "Intent" commonly means "a state of mind in which a person seeks to accomplish a given result through a course of action." Molins PLC, 48 F.3d at 1180 (citing Black's Law Dictionary at 810 (6th ed. 1990)). "Intent need not be proven by direct evidence; it is most often proven by a showing of acts, the natural consequences of which are presumably intended by the actor." Id. "For example, intent may be inferred where a patent applicant knew, or should have known, that withheld information would be material to the PTO's consideration of the patent application." Critikon, Inc. v. Becton Dickinson Vascular Access, Inc., 120 F.3d 1253, 1256 (Fed. Cir. 1997). The intent of the applicant however, must be to deceive, not simply to withhold the reference. Dayco Prod., 329 F.2d at 1367. "Intent to deceive cannot be inferred simply from the decision to withhold the reference where the reasons given for the withholding are plausible." Id.

103. "It is not inequitable conduct to omit telling

the patent examiner information that the applicant in good faith believes is not material to patentability." Allied Colloids, Inc. v. Am. Cyanamid Co., 64 F.3d 1570, 1578 (Fed. Cir. 1995). Disclosure of relevant prior art to the PTO during the course of another, subsequent patent prosecution "has no bearing on whether [the patentee] acted with deceptive intent during prosecution of the" application at issue. Li Second Family L.P., 231 F.3d at 1381.

104. A finding of inequitable conduct is "an equitable determination," therefore, "it is committed to the discretion of the trial court." Monon Corp., 239 F.3d at 1261.

105. Bayer contends the following acts and omissions constitute inequitable conduct: (1) Dr. Housey failed to disclose that status of Drs. Weinstein and Hsiao as co-inventors; (2) Dr. Housey misrepresented the role of other researchers in the conducting of certain experiments; (3) Dr. Housey misrepresented that he actually performed the soft agar experiments pertaining to table 3; (4) Dr. Housey withheld material prior art; and (5) Dr. Housey failed to disclose Columbia's potential ownership interests.

106. Housey, in response, contends: (1) the contributions of Drs. Weinstein and Hsiao were acknowledged through disclosure of the Cell Paper; (2) Drs. Weinstein and Hsiao do not claim to be co-inventors of the invention as Dr.

Housey subjectively understood it to be; (3) the prior art references cited by Bayer are not material to the invention as Dr. Housey understood it; and (4) that overwhelming evidence exists that the experiments reported in the suit were performed by Dr. Housey.

107. As an initial matter, there is one material fact that is not disputed between the parties. It was Dr. Housey's clear intent to conceal his work from his colleagues. Although engaged in what might be groundbreaking research and in a laboratory which by all accounts was highly collegial and collaborative, Dr. Housey surreptitiously prepared and filed the '281 patent application. It is against this backdrop that the court must weigh and evaluate the evidence and credibility of the witnesses.

108. **Acknowledgment of Work and Contributions of Other Scientists.** There is no dispute that the results reflected in figures 4, 5, and 7 and table 2 of the '281 patent are identical to those reported in the Cell Paper. Dr. Housey's own testimony indicates that he did not unilaterally conduct these experiments[17] and the court finds that Dr. Housey did not

[17]In reference to figure 7, which illustrates photographs of cell lines grown in soft agar, Dr. Housey states: "This was principally work where Dr. Hsiao plated out the cells and soft agar and we both looked at them over the course of several weeks." (D.I. 283 at 1034)

unilaterally conduct the experimental steps detailed in the '281 patent with respect to figures 4, 5, and 7, and table 2. While a patent applicant may rely upon the work of others in framing his invention and such reliance may not give rise to a claim of co-inventorship, the duty of candor requires that the use of another's work be fully disclosed. Consequently, the court finds that omission of the role of Dr. Housey's colleagues in the performance of these experiments is material.

109. **Absence of Primary Date Pertaining to Soft Agar Experiments.** Of particular interest to the court is the absence of any written records that substantiate Dr. Housey's testimony with respect to the soft agar experiments reported in table 3. It is indisputable that table 3 is highly material as it was specifically relied upon in overcoming the examiner's rejection. The only evidence in the record that Dr. Housey performed these experiments is Dr. Housey's testimony. In support of the absence of any corroborating written records, Dr. Housey describes a scientific method whereby he employs a calculator and attached printer so that he may conduct the experiment without the use of a traditional laboratory notebook. He fails, however, to point to a single calculator printout which might substantiate his version of the events.

110. The law does not require that a scientist record

his observations and findings in a particular medium and fashion. The failure to have handwritten primary data, therefore, is not, by itself, dispositive of whether the reported experiments occurred. In this case, however, the absence of primary data is illuminating when Dr. Housey's own meticulous habits in the laboratory are taken into consideration. Viewed in that context, the absence of primary date to corroborate his testimony is revealing.

111. **Withholding of Material Prior Art.** The court finds that the Hsiao 1986 reference would have been material to a reasonable patent examiner. Hsiao 1986, although focused on oncogene complementation, may suggest that Dr. Housey's specifications did not enable the direct interaction limitation in his claim.[18]

112. The court also concludes that Uehara 1985 would have been material to a reasonable patent examiner. The Uehara 1985 authors attempted to determine direct interaction between an agent and a target protein. While Uehara 1985 may have been initially unsuccessful, that does not negate its materiality to the patent examiner.

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[18]In light of Bayer's admission before the European Patent Office that Hsiao 1986 did not disclose a complete screening assay, it is plausible that Dr. Housey's decision to withhold Hsiao 1986 was based on an erroneous view that the reference was immaterial and not with the requisite intent to deceive.

113. The record also indicates that Dr. Housey's patent counsel and more than one licensee brought these prior art references to Dr. Housey's attention following his initial filing. The duty of candor includes the duty to supplement the application where necessary. 37 C.F.R. § 1.56(a) ("The duty to disclose information exists with respect to each pending claim until the claim is cancelled or withdrawn from consideration, or the application becomes abandoned."). The law requires that applicants err in favor of disclosure. See Brasseler, U.S.A. I, L.P. v. Stryker Sales Corp., 267 F.3d 1370, 1386 (Fed. Cir. 2001) ("[W]here the materiality of the information is uncertain, disclosure is required."). Even giving credit to Dr. Housey's testimony that he believed his invention to have a direct interaction limitation and that these references, therefore, were not material, the raising of these references by third parties put Housey on notice that these prior art references may be material.[19] The court finds, therefore, that Housey knowingly withheld this prior art.

114. **Potential Ownership Interests.** Bayer asserts that Dr. Housey should have disclosed the potential ownership

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[19]Ironically, had Dr. Housey disclosed these prior art references and explained to the examiner the asserted distinctions, the prosecution might have resulted in a claim construction more consistent with Dr. Housey's alleged interpretation.

interests of Columbia University and the United States Government. The court is not persuaded that failure to disclose potential ownership interests by itself is relevant to an inequitable conduct inquiry. The gravity of a finding of inequitable conduct demands that it be narrowly drawn to issues directly bearing on whether the invention itself is patentable. See Florida State Bd. of Educ., 333 F.3d at 1344. Its purpose is to insure candor and good faith by applicants. See Fox Indus., 922 F.2d at 803. The ownership rights therein, while important, do not bear on the whether the invention fulfills the statutory requirements. Nonetheless, Dr. Housey's conduct in this regard reenforces the pattern of concealment and possible deception which has emerged from the record.

115. **Dr. Housey's Credibility.** In viewing the record and the conduct of Dr. Housey as a whole, the court concludes that there is a pattern of behavior inconsistent with the duty of candor which undermines the credibility of Dr. Housey's testimony. See LaBounty Mfg., Inc. v. U.S. Intern. Trade Com'n, 958 F.2d 1066, 1070 (Fed. Cir. 1992)("An equitable judgment must be made that, in light of all the particular circumstances, the conduct of the patentee is so culpable that its patent should not be enforced."). Dr. Housey actively concealed his work from his colleagues. Dr. Housey failed to provide in his application clear acknowledgment of the contributions of his colleagues.

Finally, Dr. Housey knowingly withheld material prior art. While these facts and findings individually may not rise to the level of clear and convincing evidence of inequitable conduct if viewed in isolation, they do substantially undermine the credibility of Dr. Housey as a witness.

116. The credibility of Dr. Housey is a central, if not dispositive, issue in this case. The sole evidence that the soft agar experiments reported in table 3 were conducted is the uncorroborated testimony of Dr. Housey. As the court finds that Dr. Housey's testimony is not credible, this compels the conclusion that Bayer has proven by clear and convincing evidence that the soft agar experiments were not performed.

117. Having found that Dr. Housey did not perform the soft agar experiments in table 3, the court has no alternative but to conclude that Dr. Housey's conduct amounts to inequitable conduct. Intentionally misrepresenting key experiments to the PTO is conduct which cannot be explained, defended nor excused. Consequently, the court concludes that the '281 patent, and those patents which are continuations thereof, are unenforceable.[20]

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[20]On November 18, 2003, Housey filed a motion to supplement the record with the decision of the Regional Court of Düsseldorf pertaining to Housey's European patent. (D.I. 303) Without reaching the merits of that motion, the court concludes that Housey's motion to supplement is moot.